WEST Search History

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DATE: Wednesday, April 26, 2006

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	DB=PGPB; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L3	L2	0
	DB=USPT,USOC,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L2	VEGFR2 same (calcium or ca)	10
	DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L1	VEGFR2 same (calcium or ca)	10

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 10 of 10 returned.

☐ 1. Document ID: US 6887468 B1

L1: Entry 1 of 10 File: USPT May 3, 2005

US-PAT-NO: 6887468
DOCUMENT-IDENTIFIER: US 6887468 B1

TITLE: Antibody kits for selectively inhibiting VEGF

DATE-ISSUED: May 3, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorpe; Philip E.	Dallas	TX		
Brekken; Rolf A.	Seattle	WA		

US-CL-CURRENT: 424/130.1; 424/139.1, 424/143.1, 424/145.1, 530/388.1

ABSTRACT:

Disclosed are antibodies that specifically inhibit VEGF binding to only one (VEGFR2) of the two VEGF receptors. The antibodies effectively inhibit angiogenesis and induce tumor regression, and yet have improved safety due to their specificity. The present invention thus provides new antibody-based compositions, methods and combined protocols for treating cancer and other angiogenic diseases. Advantageous immunoconjugate and prodrug compositions and methods using the new VEGF-specific antibodies are also provided.

55 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

☐ 2. Document ID: US 6703020 B1

L1: Entry 2 of 10 File: USPT Mar 9, 2004

US-PAT-NO: 6703020
DOCUMENT-IDENTIFIER: US 6703020 B1
** See image for Certificate of Correction **

TITLE: Antibody conjugate methods for selectively inhibiting VEGF

DATE-ISSUED: March 9, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorpe; Philip E.	Dallas	TX		
Brekken; Rolf A.	Seattle	WA		

US-CL-CURRENT: 424/178.1; 424/130.1, 424/145.1, 424/156.1, 424/158.1, 530/387.1, 530/388.2

ABSTRACT:

Disclosed are antibodies that specifically inhibit VEGF binding to only one (VEGFR2) of the two VEGF receptors. The antibodies effectively inhibit angiogenesis and induce tumor regression, and yet have improved safety due to their specificity. The present invention thus provides new antibody-based compositions, methods and combined protocols for treating cancer and other angiogenic diseases. Advantageous immunoconjugate and prodrug compositions.

49 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 4

☐ 3. Document ID: US 6676941 B2

L1: Entry 3 of 10 File: USPT Jan 13, 2004

US-PAT-NO: 6676941
DOCUMENT-IDENTIFIER: US 6676941 B2
**** See image for Certificate of Correction ****

TITLE: Antibody conjugate formulations for selectively inhibiting VEGF

DATE-ISSUED: January 13, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorpe; Philip E.	Dallas	TX		
Brekken; Rolf A.	Seattle	WA		

US-CL-CURRENT: 424/178.1; 424/1.49, 424/1.53, 424/130.1, 424/179.1, 424/181.1, 424/183.1, 424/193.1, 424/195.11, 424/9.3 , 424/9.34, 424/9.6, 435/7.1, 435/7.2, 435/7.3, 435/810, 530/391.1, 530/391.3, 530/391.5, 530/391.7, 530/391.9

ABSTRACT:

Disclosed are antibodies that specifically inhibit VEGF binding to only one (VEGFR2) of the two VEGF receptors. The antibodies effectively inhibit angiogenesis and induce tumor regression, and yet have improved safety due to their specificity. The present invention thus provides new antibody-based compositions, methods and

combined protocols for treating cancer and other angiogenic diseases. Advantageous immunoconjugate and prodrug compositions and methods using the new VEGF-specific antibodies are also provided.

68 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. D

☐ 4. Document ID: US 6610484 B1

L1: Entry 4 of 10 File: USPT Aug 26, 2003

US-PAT-NO: 6610484
DOCUMENT-IDENTIFIER: US 6610484 B1
**** See image for Certificate of Correction ****

TITLE: Identifying material from a breast duct

DATE-ISSUED: August 26, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hung; David T.	Belmont	CA		

US-CL-CURRENT: 435/6; 435/7.1, 435/7.2, 435/7.23, 435/7.92, 436/501, 436/503, 436/504, 436/63, 436/64

ABSTRACT:

Methods and systems for identifying material from a breast duct using one or more markers that can be identified in ductal fluid retrieved from the breast are provided.

23 Claims, 0 Drawing figures
Exemplary Claim Number: 1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. D

☐ 5. Document ID: US 6524583 B1

L1: Entry 5 of 10 File: USPT Feb 25, 2003

US-PAT-NO: 6524583
DOCUMENT-IDENTIFIER: US 6524583 B1
**** See image for Certificate of Correction ****

TITLE: Antibody methods for selectively inhibiting VEGF

DATE-ISSUED: February 25, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorpe; Philip E.	Dallas	TX		
Brekken; Rolf A.	Seattle	WA		

US-CL-CURRENT: 424/145.1; 424/133.1, 424/135.1, 424/141.1, 530/387.1, 530/388.1, 530/388.15, 530/388.25, 530/809, 530/864, 530/865, 530/866

ABSTRACT:

Disclosed are antibodies that specifically inhibit VEGF binding to only one (VEGFR2) of the two VEGF receptors. The antibodies effectively inhibit angiogenesis and induce tumor regression, and yet have improved safety due to their specificity. The present invention thus provides new antibody-based compositions, methods and combined protocols for treating cancer and other angiogenic diseases. Advantageous immunoconjugate and prodrug compositions and methods using the new VEGF-specific antibodies are also provided.

40 Claims, 7 Drawing figures
Exemplary Claim Number: 1,4
Number of Drawing Sheets: 4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. D

☐ 6. Document ID: US 6416758 B1

L1: Entry 6 of 10 File: USPT Jul 9, 2002

US-PAT-NO: 6416758
DOCUMENT-IDENTIFIER: US 6416758 B1
**** See image for Certificate of Correction ****

TITLE: Antibody conjugate kits for selectively inhibiting VEGF

DATE-ISSUED: July 9, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorpe; Philip E.	Dallas	TX		
Brekken; Rolf A.	Seattle	WA		

US-CL-CURRENT: 424/145.1; 424/1.49, 424/1.53, 424/1.69, 424/133.1, 424/134.1, 424/135.1, 424/141.1, 424/142.1, 424/178.1 , 424/179.1, 424/181.1, 424/183.1, 424/195.11, 424/9.2, 424/9.3, 435/69.1, 435/69.6, 435/69.7, 435/7.23, 435/70.21, 435/810, 530/387.3, 530/388.1, 530/388.15, 530/388.24, 530/391.3, 530/391.7, 530/391.9

ABSTRACT:

Disclosed are antibodies that specifically inhibit VEGF binding to only one

(VEGFR2) of the two VEGF receptors. The antibodies effectively inhibit angiogenesis and induce tumor regression, and yet have improved safety due to their specificity. The present invention thus provides new antibody-based compositions, methods and combined protocols for treating cancer and other angiogenic diseases. Advantageous immunoconjugate and prodrug compositions.

50 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 4

☐ 7. Document ID: US 6413228 B1

L1: Entry 7 of 10 File: USPT Jul 2, 2002

US-PAT-NO: 6413228
DOCUMENT-IDENTIFIER: US 6413228 B1

TITLE: Devices, methods and systems for collecting material from a breast duct

DATE-ISSUED: July 2, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Hung; David	Belmont	CA		
Ken; Christopher G. M.	San Mateo	CA		
He; Xuanmin	Palo Alto	CA		
Olsen; Phillip M.	Mountain View	CA		
Nikolchev; Julian	Portola Valley	CA		
O'Leary; Shawn	San Jose	CA		
Sayavong; Pam	Newark	CA		

US-CL-CURRENT: 600/562; 435/7.23, 600/573, 604/28

ABSTRACT:

The invention provides methods, devices and systems for collecting breast ductal fluid comprising cellular material and other useful markers for analysis. The methods typically comprise access of at least one breast duct and collecting materials from that duct separate from all other ducts in the breast. The devices comprise ductal access devices that provide the opportunity to collect fluid from a single duct separate from all the other ducts in the breast. The systems employ the methods and devices that used together provide systems for analysis of a breast condition in a patient specific to accessed breast ducts. The methods, devices and systems are particularly useful for indentification of breast precancer or cancer in patient.

101 Claims, 18 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 12

☐ 8. Document ID: US 6342221 B1

L1: Entry 8 of 10 File: USPT Jan 29, 2002

US-PAT-NO: 6342221
DOCUMENT-IDENTIFIER: US 6342221 B1
** See image for Certificate of Correction **

TITLE: Antibody conjugate compositions for selectively inhibiting VEGF

DATE-ISSUED: January 29, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorpe; Philip E.	Dallas	TX		
Brekken; Rolf A.	Seattle	WA		

US-CL-CURRENT: 424/178.1; 424/1.49, 424/1.53, 424/130.1, 424/179.1, 424/181.1, 424/183.1, 424/193.1, 424/195.11, 424/9.3 , 424/9.34, 424/9.6, 435/69.1, 435/7.1, 435/7.21, 435/7.23, 435/70.21, 435/810, 530/391.1, 530/391.3, 530/391.5, 530/391.7, 530/391.9

ABSTRACT:

Disclosed are antibodies that specifically inhibit VEGF binding to only one (VEGFR2) of the two VEGF receptors. The antibodies effectively inhibit angiogenesis and induce tumor regression, and yet have improved safety due to their specificity. The present invention thus provides new antibody-based compositions, methods and combined protocols for treating cancer and other angiogenic diseases. Advantageous immunoconjugate and prodrug compositions and methods using the new VEGF-specific antibodies are also provided.

68 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 9. Document ID: US 6342219 B1

L1: Entry 9 of 10 File: USPT Jan 29, 2002

US-PAT-NO: 6342219
DOCUMENT-IDENTIFIER: US 6342219 B1

TITLE: Antibody compositions for selectively inhibiting VEGF

DATE-ISSUED: January 29, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Thorpe; Philip E.	Dallas	TX		
Brekken; Rolf A.	Seattle	WA		

US-CL-CURRENT: 424/145.1; 424/133.1, 424/134.1, 424/135.1, 424/141.1, 424/142.1, 424/143.1, 435/335, 435/69.1, 435/810, 530/387.1, 530/387.3, 530/388.1, 530/388.15, 530/388.23, 530/391.1, 530/391.3, 530/391.5, 530/391.7, 530/809, 530/864, 530/865, 530/866

ABSTRACT:

Disclosed are antibodies that specifically inhibit VEGF binding to only one (VEGFR2) of the two VEGF receptors. The antibodies effectively inhibit angiogenesis and induce tumor regression, and yet have improved safety due to their specificity. The present invention thus provides new antibody-based compositions, methods and combined protocols for treating cancer and other angiogenic diseases. Advantageous immunoconjugate and prodrug compositions and methods using the new VEGF-specific antibodies are also provided.

50 Claims, 7 Drawing figures
Exemplary Claim Number: 20
Number of Drawing Sheets: 4

☐ 10. Document ID: US 6077673 A

L1: Entry 10 of 10 File: USPT Jun 20, 2000

US-PAT-NO: 6077673
DOCUMENT-IDENTIFIER: US 6077673 A

TITLE: Mouse arrays and kits comprising the same

DATE-ISSUED: June 20, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Chenchik; Alex	Palo Alto	CA		
Lukashev; Matvey	Newton	MA		

US-CL-CURRENT: 435/6; 422/68.1, 435/283.1, 435/285.1, 435/286.1, 435/286.2, 435/287.1, 435/287.2, 435/287.7, 435/287.9, 435/289.1, 435/299.1

ABSTRACT:

Mouse arrays and methods for their use are provided. The subject arrays include a plurality of polynucleotide spots, each of which is made up of a polynucleotide probe composition of unique polynucleotides corresponding to a key mouse gene. The subject arrays find use in hybridization assays, particularly in assays for the identification of differential gene expression of key mouse genes of interest.

17 Claims, 0 Drawing figures
Exemplary Claim Number: 1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. D

Clear | Generate Collection | Print | Fwd Refs | Bkwd Refs | Generate OACS

Terms	Documents
VEGFR2 same (calcium or ca)	10

Display Format: REV Change Format

Previous Page Next Page Go to Doc#

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FILE 'HOME' ENTERED AT 19:17:23 ON 26 APR 2006

=> file .nash

=> s vegfr2 and (calcium ion or calcium flux)
L1            1 FILE MEDLINE
L2            2 FILE CAPLUS
L3            1 FILE SCISEARCH
L4            1 FILE LIFESCI
L5            1 FILE BIOSIS
L6            4 FILE EMBASE

TOTAL FOR ALL FILES
L7           10 VEGFR2 AND (CALCIUM ION OR CALCIUM FLUX)

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8            5 DUP REM L7 (5 DUPLICATES REMOVED)

=> d ibib abs 1-5

L8   ANSWER 1 OF 5     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:   2005420268     MEDLINE Full-text
DOCUMENT NUMBER:    PubMed ID: 15980434
TITLE:              Inhibition of vascular permeability factor/vascular
                    endothelial growth factor-mediated angiogenesis by the
                    Kruppel-like factor KLF2.
AUTHOR:             Bhattacharya Resham; Senbanerjee Sucharita; Lin Zhiyong;
                    Mir Samy; Hamik Anne; Wang Ping; Mukherjee Priyabrata;
                    Mukhopadhyay Debabrata; Jain Mukesh K
CORPORATE SOURCE:   Program in Cardiovascular Transcriptional Biology,
                    Cardiovascular Division, Brigham and Women's Hospital,
                    Harvard Medical School, Boston, MA 02115, USA.
CONTRACT NUMBER:    CA78383 (NCI)
                    HL-72952 (NHLBI)
                    HL-76754 (NHLBI)
                    HL072178 (NHLBI)
                    HL69477 (NHLBI)
                    HL70567 (NHLBI)
                    HL75427 (NHLBI)
SOURCE:             The Journal of biological chemistry, (2005 Aug 12) Vol.
                    280, No. 32, pp. 28848-51.  Electronic Publication:
                    2005-06-26.
                    Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200509
ENTRY DATE:         Entered STN: 9 Aug 2005
                    Last Updated on STN: 23 Sep 2005
                    Entered Medline: 22 Sep 2005
AB     The Kruppel-like factor KLF2 was recently identified as a novel regulator of endothelial
       pro-inflammatory and pro-thrombotic function.  Here it is shown that overexpression of
       KLF2 potently inhibits vascular permeability factor/vascular endothelial growth factor
       (VEGF-A)-mediated angiogenesis and tissue edema in the nude ear mouse model of
       angiogenesis.  In vitro, KLF2 expression retards VEGF-mediated calcium flux, proliferation
       and induction of pro-inflammatory factors in endothelial cells.  This effect is due to a
       potent inhibition of VEGFR2/KDR expression and promoter activity.  These observations
       identify KLF2 as a regulator of VEGFR2/KDR and provide a foundation for novel approaches
       to regulate angiogenesis.


L8   ANSWER 2 OF 5  EMBASE  COPYRIGHT (c) 2006 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:   2004357975  EMBASE  Full-text
TITLE:              Vascular endothelial growth factor (VEGF)-D and VEGF-A
                    differentially regulate KDR-mediated signaling and
                    biological function in vascular endothelial cells.
```

AUTHOR: Jia H.; Bagherzadeh A.; Bicknell R.; Duchen M.R.; Liu D.; Zachary I.
CORPORATE SOURCE: I. Zachary, Dept. of Medicine, Rayne Institute, University College London, 5 University St., London WC1E 6JJ, United Kingdom. i.zachary@ucl.ac.uk
SOURCE: Journal of Biological Chemistry, (20 Aug 2004) Vol. 279, No. 34, pp. 36148-36157. .
Refs: 36
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Sep 2004
Last Updated on STN: 16 Sep 2004

AB Vascular endothelial growth factor (VEGF)-D binds to VEGF receptors (VEGFR) VEGFR2/KDR and VEGFR3/ Flt4, but the signaling mechanisms mediating its biological activities in endothelial cells are poorly understood. Here we investigated the mechanism of action of VEGF-D, and we compared the signaling pathways and biological responses induced by VEGF-D and VEGF-A in endothelial cells. VEGF-D induced KDR and phospholipase C-γ tyrosine phosphorylation more slowly and less effectively than VEGF-A at early times but had a more sustained effect and was as effective as VEGF-A after 60 min. VEGF-D activated extracellular signal-regulated protein kinases 1 and 2 with similar efficacy but slower kinetics compared with VEGF-A, and this effect was blocked by inhibitors of protein kinase C and mitogen-activated protein kinase kinase. In contrast to VEGF-A, VEGF-D weakly stimulated prostacyclin production and gene expression, had little effect on cell proliferation, and stimulated a smaller and more transient increase in intracellular [Ca(2+)]. VEGF-D induced strong but more transient phosphatidylinositol 3-kinase (PI3K)-mediated Akt activation and increased PI3K-dependent endothelial nitric-oxide synthase phosphorylation and cell survival more weakly. VEGF-D stimulated chemotaxis via a PI3K/Akt- and endothelial nitric-oxide synthase-dependent pathway, enhanced protein kinase C- and PI3K-dependent endothelial tubulogenesis, and stimulated angiogenesis in a mouse sponge implant model less effectively than VEGF-A. VEGF-D-induced signaling and biological effects were blocked by the KDR inhibitor SU5614. The finding that differential KDR activation by VEGF-A and VEGF-D has distinct consequences for endothelial signaling and function has important implications for understanding how multiple ligands for the same VEGF receptors can generate ligand-specific biological responses.

L8 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:323276 CAPLUS Full-text
DOCUMENT NUMBER: 139:95574
TITLE: Recruitment and activation of phospholipase Cγ1 by vascular endothelial growth factor receptor-2 are required for tubulogenesis and differentiation of endothelial cells
AUTHOR(S): Meyer, Rosana D.; Latz, Catharina; Rahimi, Nader
CORPORATE SOURCE: Departments of Ophthalmology and Biochemistry, Boston University School of Medicine, Boston, MA, 02118, USA
SOURCE: Journal of Biological Chemistry (2003), 278(18), 16347-16355
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Vascular endothelial growth factor-mediated angiogenic signal transduction relay is achieved by coordinated induction of endothelial cell proliferation, migration, and differentiation. These complex cellular processes are most likely controlled by activation of both cooperative and antagonistic signals by vascular endothelial growth factor receptors (VEGFRs). Here, the authors investigated the contribution of tyrosine-phosphorylated residues of VEGFR-2/fetal liver kinase-1 to endothelial cell proliferation and differentiation and activation of signaling proteins. Mutation of tyrosine 1006 of VEGFR-2 to phenylalanine severely impaired the ability of this receptor to stimulate endothelial cell differentiation and tubulogenesis. Paradoxically, the mutant receptor stimulated endothelial cell proliferation far better than the wild-type receptor. Further anal. showed that tyrosine 1006 is responsible for phospholipase Cγ1 (PLCγ1) activation and intracellular calcium release in endothelial cells. Activation of PLCγ1 was selectively

mediated by tyrosine 1006. Mutation of tyrosines 799, 820, 949, 994, 1080, 1173, and 1221 had no measurable effect on the ability of VEGFR-2 to stimulate PLCγ1 activation. Association of VEGFR-2 with PLCγ1 was mainly established between tyrosine 1006 and the C-terminal SH2 domain of PLCγ1 in vitro and in vivo. Taken together, the results indicate that phosphorylation of tyrosine 1006 is essential for VEGFR-2-mediated PLCγ1 activation, calcium flux, and cell differentiation. More importantly, VEGFR-2-mediated endothelial cell proliferation is inversely correlated with the ability of VEGFR-2 to associate with and activate PLCγ1.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 4 OF 5 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003200648 EMBASE Full-text
TITLE: Functional evidence that vascular endothelial growth factor may act as an autocrine factor on human podocytes.
AUTHOR: Foster R.R.; Hole R.; Anderson K.; Satchell S.C.; Coward R.J.; Mathieson P.W.; Gillatt D.A.; Saleem M.A.; Bates D.O.; Harper S.J.
CORPORATE SOURCE: D.O. Bates, Microvascular Research Laboratories, Dept. of Physiology, Univ. of Bristol, Southwell St., Bristol BS2 8EJ, United Kingdom. Dave.Bates@bristol.ac.uk
SOURCE: American Journal of Physiology - Renal Physiology, (1 Jun 2003) Vol. 284, No. 6 53-6, pp. F1263-F1273. .
Refs: 41
ISSN: 0363-6127 CODEN: AJPPFK
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
028 Urology and Nephrology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 5 Jun 2003
Last Updated on STN: 5 Jun 2003

AB Vascular endothelial growth factor (VEGF) is expressed by renal glomerular epithelial cells (podocytes) and is thought to be protective against nephrotoxic agents. VEGF has been shown to be an autocrine survival factor in neuropilin-1-positive, VEGF receptor-negative breast carcinoma cells. Normal human podocytes are also known to express neuropilin-1, VEGF, and are VEGFR2 negative. Here, we investigated whether a similar VEGF autocrine loop may exist in podocytes. Podocyte cytosolic calcium concentration ([Ca(2+)](i)) was analyzed in primary cultured and conditionally immortalized podocytes using ratiometric fluorescence measurement. Cytotoxicity was determined by lactate dehydrogenase assay, proliferation by [(3)H]-thymidine incorporation, and cell counts by hemocytometric assay. VEGF decreased [Ca(2+)](i) in primary podocytes (from 179 ± 36 to 121 ± 25 nM, P < 0.05) and conditionally immortalized podocytes (from 95 ± 10 to 66 ± 8 nM, P < 0.02) in the absence of extracellular calcium. The type III receptor tyrosine-kinase inhibitor PTK787/ ZK222584 abolished this reduction. VEGF increased podocyte [(3)H]-thymidine incorporation (3,349 ± 283 cpm, control 2,364 ± 301 cpm, P < 0.05) and cell number (4.5 ± 0.7 x 10(4)/ml, control 2.6 ± 0.5 x 10(4)/ml, P < 0.05) and decreased cytotoxicity (5.9 ± 0.7%, control 12 ± 3%, P < 0.05), whereas a monoclonal antibody to VEGF increased cytotoxicity. Electron microscopy of normal human glomeruli demonstrated that the glomerular VEGF is mostly podocyte cell membrane associated. These results indicate that one of the functions of VEGF secreted from podocytes may be to act as an autocrine factor on calcium homeostasis and cell survival.

L8 ANSWER 5 OF 5 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2001365795 EMBASE Full-text
TITLE: HIV-Tat dependent chemotaxis and invasion, key aspects of Tat mediated pathogenesis.
AUTHOR: Vene R.; Benelli R.; Noonan D.M.; Albini A.
CORPORATE SOURCE: Dr. A. Albini, Molecular Biology Laboratory, Natl. Institute for Cancer Research, Largo Rosanna Benzi 10, 16132 Genova, Italy. albini@cba.unige.it
SOURCE: Clinical and Experimental Metastasis, (2001) Vol. 18, No. 7, pp. 533-538. .
Refs: 64
ISSN: 0262-0898 CODEN: CEXMD2
COUNTRY: Netherlands

DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 016 Cancer
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 2 Nov 2001
Last Updated on STN: 2 Nov 2001

AB Extracellular Tat acts as a pleiotropic molecule inducing several biological effects on different target cells. Tat stimulates the chemotaxis of numerous cell types and it appears to have oncogenic activities, including acting as a co-factor for Kaposi's sarcoma. The Tat protein has been shown to bind integrins through an RGD amino acid motif. Tat is an angiogenic factor able to induce the migration and invasion of endothelial and KS cells through the interaction of its basic domain with the VEGF receptor VEGFR2 (Flk-1/KDR). We have also found that Tat is able to mimic chemokines, activating monocyte migration through the 'chemokine like' cysteine-core domain. Tat is a chemoattractant for dendritic cells, and both the RGD and basic domains appear to be involved in this response. In a recent study we demonstrated that Tat is chemotactic for PMN and induces Ca(2+) mobilization in vitro. Experiments using synthetic peptides showed that Tat activities on PMN are mediated by the 'chemokine like' region. Finally Tat is also able to induce B cell chemotaxis, while its activity on helper T cells has not yet been clarified. Here we review data on Tat-dependent chemotaxis and discuss the possible implications in Tat mediated pathogenesis.

=> log y